repudiated or substantiated, the sooner this guide is properly sought for the sooner will an important advance be made in this branch of medicine.

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SOME FACTS AND FALLACIES CONCERNING ABDOMINAL ADHESIONS AND BANDS.

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WITH the continued development of abdominal surgery there has been an increasing appreciation of the importance of the normal activities and pathological conditions of the peritoneum. The recognition of gross lesions of this structure, such as the more evident forms of active peritonitis and the resultant inflammatory adhesions, antedates by far our knowledge of the conditions giving rise to them. The demonstration of the bacterial origin of peritoneal lesions and subsequent investigations as to the causation of such bacterial invasions have placed our knowledge of the more acute forms of peritoneal disease upon a firm basis.

A far more difficult problem presents itself when we consider peritoneal adhesions and bands for which it is impossible to ascertain a definite antecedent acute inflammatory lesion. The recognition of such structures is not a recent one. It has been stated that Virchow, in 1858, was the first to describe these structures more in detail, but at that time, of course, there was no basis for a correct opinion concerning their causation. For a long time after the beginning of abdominal surgery upon a larger scale the avoidance and treatment of acute peritonitis engrossed the attention of operators. Further and extended observation of early cases led to information concerning postoperative and postinflammatory adhesions and to efforts to determine their cause and to attempts at their prevention. A still later development of the matter was an endeavor to understand the symptomatology and diagnosis of abdominal adhesions as apart from other lesions.

Finally the work of such men as Jonnescu, Lane, and Treves, several decades ago, called the attention of surgeons to the existence of various folds, bands, and membranes in relation to portions of the gastro-intestinal tract and to their possible importance. It remained for J. N. Jackson, in 1909, to bring to a definite standing in our minds the significance of certain pericolic structures or membranes, and following him many investigators and clinicians have endeavored to place upon a firmer basis our knowledge of this subject.

A great deal of confusion which exists concerning all these adhesions, bands, membranes, etc., may be obviated by the clearer grasp of certain fundamentals, and an effort to distinguish between various groups and varieties of these structures, instead of attempting to make one theory as to their causation explain every case.

All abdominal adhesions and bands may be grouped into two classes:

1. Those adhesions and similar structures whose character and location make it evident that they must have been the result of a definite attack or attacks of peritonitis of more or less severity.

2. Those structures in which some doubt may be entertained as to their formation as the result of a distinct peritonitis.

The former class gives us more data upon which to form a conclusion; the latter is the one about which the discussion among surgeons at present centers. Jackson himself has stated that he is in the dark as to the causation of the membrane named after him or Jonnescu; others have been more definite in statements as to their opinions.

Membranes and adhesions not frankly the results of a peritonitis may be again subdivided into:

(*a*) Structures whose character and location make it possible that they may be the results of fetal maldevelopment.

(*b*) Similar membranes in localities in which this causation is not so plausible.

The explanation of the existence of both of these types has been attempted upon numerous grounds. Briefly, they are:

1. The attempt to explain all or most of these formations on the basis of failure of the rotation or descent of the gut or an anomalous growth of the peritoneum and mesentery in the presence of normal rotation. While there may be some basis for this assumption in certain cases, it has appeared more than a coincidence to us that these bands or veils with their attendant symptoms should be so often localized at or near that portion of the gut where we are most likely to have lesions of an inflammatory nature. Nor is it a counterbalancing fact that there is not to be found the remaining proof of such an inflammation.

Eastman, in experiments on rabbits, by artificially producing coprostasis, has succeeded in producing a low-grade peritonitis and subsequent formation of membranes. Cheever is one of those who dissent most strongly from this view of the matter, and his dissections somewhat strengthen his position. Nevertheless the experiments mentioned and those of Murphy and others on intestinal obstrucion lead us to the belief that in many cases a migration of bacteria through the intestinal wall can and does give rise to diffuse peritoneal inflammation of a low grade which results in the formation of veils, membranes, and bands.

It is a remarkable fact that these structures are most often found in adult life and at that time give rise to symptoms. Is not this also an evidence that an early underlying cause gives rise to conditions which cause their symptoms later, when the membrane or other structure becomes of such extent as to seriously interfere with the activity of the gut?

Granting at once that certain of the structures might possibly be the results of causes unassociated with inflammatory changes, there is no evidence that this is the case, and it is far more reasonable to suppose them the results of an inflammation until the reverse is proven.

2. Mr. Arbuthnot Lane and others (Fagge, etc.) have attempted to explain peritoneal bands as "crystallization of lines of force," and the result of stress upon normal structures causing them to be altered. There is no doubt that malposition of abdominal viscera must bring with it a malposition of their surrounding structures and coverings, but the whole of Mr. Lane's explanation of these conditions is surrounded by a haze of theory, argument, and supposition unsupported by facts. His theories are curious and interesting, but entirely unconvincing; plausible on first reading but not susceptible of close reasoning. Some of his views and statements do not bear analysis at all; their true inward meaning must be known only to Mr. Lane himself. To explain a definite and often pathological structure upon such vague and fanciful grounds does not tend to clarify the situation; it only introduces an additional mystery to a difficult subject.

3. In contradistinction to these views there are those who would explain practically all of these new or malformations of the peritoneum as the results of inflammation. It is a matter of no great consequence that many of these adhesions or bands are or seem to be congenital. The evidences of antenatal inflammation of the peritoneum are sufficiently definite in numbers of reported cases (Doran, Kieth, Ballantyne, and Veszprenit). It is the opinion of these men that such instances of fetal peritonitis as they cite are by no means uncommon, and the examples given are those in which there was no evidence of a purely mechanical origin of the peritonitis such as a perforation. The transmission of toxins and drugs from

mother to fetus is universally acknowledged, as is the transmission of the infecting agents themselves by means of the blood or indirectly through the fetal appendages (Burnet).

Since then the existence of not infrequent antenatal peritonitis is proven, we believe that those adhesions which are found not to have been caused by a peritoneal inflammation after birth are the result of a similar condition in the fetus; that whereas many of these adhesions are congenital they are not developmental in origin. It must be understood that from such an origin we exclude those instances in which there is but an accentuation or simple enlargement of a peritoneal membrane, fold or process normally present.

In taking inflammation, acute or subacute, as a causative factor of a condition, we are dealing with a fundamental entity, a condition with the results of which we meet every day. In assuming developmental errors as causative factors of lesions evidently pathological, we can only adopt the absence of other demonstrable causes as proofs of a supposed cause.

A consideration of the opinions of various clinicians and experimenters makes evident the fact that there is no agreement concerning these bands of membranes.

Fallon even states that Jackson's membrane is always present, and is a normal structure. With this view we are not at all in agreement. Lynch, after citing certain facts to prove that Jackson's membrane is not of inflammatory origin, concludes "that the membrane described by Jackson is of embryonal origin, but it may be so altered by inflammation as to appear like an ordinary adhesion." It may be embryonal and nevertheless inflammatory, and to distinguish between a membrane of inflammatory origin—antenatal or postnatal—and a membrane merely altered by an inflammatory process appears to us to be impossible.

We think there is every reason to believe that practically all of these structures are the result of peritoneal inflammation of some kind; and, moreover, we are convinced that more extended clinical observation on the part of those who have had greater opportunities to theorize about than to observe these formations can only serve to make them agree with this opinion.

Concerning adhesions which are frankly the result of one or more attacks of peritonitis after birth a great mass of literature has been accumulated. We believe that the fact is often lost sight of that such adhesions are principally an effort of nature to effect a cure or to limit infection. The various attempts, hitherto unsuccessful, to avoid such adhesions in inflammatory cases we believe to be efforts in the wrong direction. On the other hand, in clean cases in which infection plays no part, it is essential that by minimizing handling and exposure of the viscera we avoid the formation of adhesions of no value and possibly of great harm.

In conclusion, we would merely mention the increasing recognition of the importance of abdominal adhesions, and our progress in their diagnosis is especially by means of the roentgen-ray. A less pleasing subject for our contemplation is our inability effectually to deal with such adhesions when they do exist and cause symptoms.

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THE TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM WITH INTRASPINAL INJECTIONS OF MERCURIALIZED SERUM.

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UNTIL recently the treatment of syphilis of the central nervous system was not essentially different from that of syphilis elsewhere in the body. Because of this, results in these cases have been far from satisfactory.

Since the discovery that the Spirocheta pallida are the active agents and are found viable in the tissues and exudates in the so-called parasyphilitic diseases, the tendency has been to consider these cases merely cerebrospinal syphilis and to lose sight of the finer anatomical localizations. This also is in part due to the rapid advance in our laboratory methods.

Ravaut was the first to call attention to the diagnostic importance of a meningeal reaction in the very earliest stages of syphilis, which was too slight to be detected in any other way, except by the examination of the cerebrospinal fluid. Udo Wile, in this country, has been very active in substantiating this work of Ravaut's. This can mean only one thing—that the central nervous system is involved in the early stages in the majority of cases. Systemic treatment here is usually quite efficacious, even though there may